Yu, Misook

From: Mosher, Mary
Sent: Thursday, January 23, 2003 3:25 PM
To: Yu, Misook
Subject: RE: 09/536,089

If you want to make it final, stick with the rejections made last time around, even if you can think of ways to do it better now (so stick with enablement for fragments, description for therapy, if that's what it was). Deal with the arguments by pointing out how gene therapy is different from administering a protein (intracellular vs extracellular, different uptake problems, different immunological responses, whatever is relevant). It sounds to me like they did not possess or enable a treatment method involving administering protein, if the guidance is all administering nucleic acids (and no working example).

Bad news for you - I'm totally swamped at this end of the biweek, I asked Tony to sign your cases (he's taking them tomorrow). I also have asked both SPEs to give me a sabbatical on training for about 3 biweeks, so I could deal with the festering old & ugly cases that have piled up in my office. I'm going to post a do-not-disturb sign so I can work without interruption. If I can clear out the mess in less time, I'll reopen the door early. Sorry.
MM

-----Original Message-----
From: Yu, Misook
Sent: Thursday, January 23, 2003 2:26 PM
T : Caputa, Anthony
Cc: Mosher, Mary
Subject: 09/536,089

This case is due this bi-week. The claims are drawn to method of treating cancer using TSP-2 protein and fragments. SEQ ID NO:2, 90 % sequence identity to SEQ ID NO:2, and its fragments. Two 112/1st rejections were made.

Under enablement rejection, the previous Office action says that protein chemistry is difficult to predict, therefore all of products in the claims are not enabled.

Under written description rejection, the Office action says that the spec presents evidence for treating cancer with gene therapy but does not present evidence for using the proteins or fragment.

The specification teaches only gene therapy. The amendment points to the specification with gene therapy. I would like to say the specification is not enabled for cancer treatment using the claimed product. Does the specification not showing evidence for cancer treatment go under written description? How do I proceed from here? I believe all the problem with this case were raised in this case in the previous Office action. How can I make this action final?

Examiner Misook Yu, Ph.D.
703-308-2454 (Phone)
Art Unit 1642
CM1-8E18 (Room)
CM1-8E12 (Mail Box)

WEST

Create A Case

Select?	Database	Query	Plural	Op	Thesaurus	Set Name
☑	USPT	tsp-2	YES	ADJ	ASSIGNEE	L1
☑	USPT	thrombospondin	YES	ADJ	ASSIGNEE	L2
☑	USPT	L2 and cancer	YES	ADJ	ASSIGNEE	L3
☑	USPT	L3 and thrombospondin-2	YES	ADJ	ASSIGNEE	L4
☑	USPT	thrombospondin-2	YES	ADJ	ASSIGNEE	L5
☑	USPT	5750502.pn.	YES	ADJ	ASSIGNEE	L6

Please enter the case name: 09/536087

Clear All | Reset | Create Case | Cancel

Help | Main Menu | Logout

Rules for naming Cases

- Case names can only contain alphanumeric characters including underscore (_).
- Any other special characters or punctuation characters will be automatically removed prior to saving the case.
- All white space characters will be replaced by an underscore.

1: Cancer Gene Ther 2000 Dec;7(12):1537-42 Related Articles, Links

nature publishing group

The application of an anti-angiogenic gene (thrombospondin-1) in the treatment of human prostate cancer xenografts.

Jin RJ, Kwak C, Lee SG, Lee CH, Soo CG, Park MS, Lee E, Lee SE.

Deparment of Urology, Seoul National Untiversity College of Medicine, South Korea.

Angiogenesis is a critical event for solid tumor growth and metastasis. Within a given microenvironment, the angiogenic response is determined in part by the balance between angiogenesis inducers and inhibitors. The aim of this study was to establish a thrombospondin-1 (TSP-1) (an antiangiogenic gene) expression vector, and to determine the feasibility for use of TSP-1 in prostate cancer gene therapy. The results of this study showed that pCR-TSP-1, the cloned TSP-1 expression plasmid vector, expressed the TSP-1 gene efficiently in DU145, a human prostate cancer cell line. pCR - TSP-1 did not exert any significant growth inhibitory activity on the tested cell line in vitro. However, TSP-1 overexpression inhibited the growth of DU-145 xenografts in Balb/c nude mice when directly transfected with pCR-TSP-1 in combination with a liposomal agent (DOSPER). Histological analysis showed that there were extensive areas of necrosis in the TSP-1 overexpressing tumors, whereas no necrotic foci were observed in the control tumors. Furthermore, the microvessel density was lower in the TSP-1 overexpressing tumors compared to the control tumors. These results suggest that TSP-1 may be a potentially useful gene for prostate cancer gene therapy.

PMID: 11228532 [PubMed - indexed for MEDLINE]

Display Abstract Show: 20 Sort Send to Text

i686-pc-linux-gnu Jan 7 2003 16:40:32

Gene therapy

Display Abstract Show: 20 Sort Send to Text

1: Cancer Res 2002 Apr 1;62(7):2004-12 Related Articles, Links

Systemic inhibition of tumor growth and angiogenesis by thrombospondin-2 using cell-based antiangiogenic gene therapy.

Streit M, Stephen AE, Hawighorst T, Matsuda K, Lange-Asschenfeldt B, Brown LF, Vacanti JP, Detmar M.

Cutaneous Biology Research Center and Department of Dermatology, Massachusetts General Hospital and Harvard Medical School, Charlestown 02129, USA.

Recent studies indicate that continuous administration improves the antitumoral efficacy of angiogenesis inhibitors, as compared with intermittent dosing, suggesting a potential role of gene therapy in antiangiogenic tumor therapy. We established a tissue-engineered implant system for the continuous in vivo production of thrombospondin-2 (TSP-2), a potent endogenous inhibitor of tumor growth and angiogenesis. Fibroblasts were retrovirally transduced to overexpress TSP-2 and were seeded onto biodegradable polymer scaffolds. After transplantation into the peritoneal cavity of nude mice, bioimplants maintained high levels of TSP-2 secretion over extended time periods, resulting in increased levels of circulating TSP-2. Bioimplant-generated TSP-2 potently inhibited tumor growth and angiogenesis of human squamous cell carcinomas, malignant melanomas, and Lewis lung carcinomas that were implanted at a distant site. These results provide the first proof-of-principle for the feasibility and therapeutic efficiency of systemic, cell-based antiangiogenic gene therapy using biodegradable polymer grafts for the treatment of cancer.

PMID: 11929817 [PubMed - indexed for MEDLINE]